Active immunization of the whole civilian population against tetanus in peacetime is rejected as impracticable, but it is suggested that the active immunization of children at about the age of entry to school would be both practicable and justified, especially in rural districts.

REFERENCES

- Boyd, J. S. K. (1946). *Lancet*, **1**, 113.
 Bruce, D. (1920). *J. Hyg., Camb.*, **19**, 1.
 Lassen, H. C. A. (1949). *Acta med. scand.*, **136**, Suppl. 234, p. 214.
 Long, A. P. (1948). *Amer. J. publ. Hlth.*, **38**, 485.
 — and Sartwell, P. E. (1947). *Bull. U.S. Army med. Dept.*, **7**, 371.
 Parish, H. J. (1948). *Bacterial and Virus Diseases*, p. 48. Livingstone, Edinburgh.
 Press, E. (1948). *New Engl. J. Med.*, **239**, 50.
 Ramon, G. (1950). *Le Principe des Anatoxines et ses Applications*. Masson, Paris.

SPONTANEOUS INTRAPERITONEAL RUPTURE OF THE BLADDER IN THE PUERPERIUM

BY

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In most cases in which the bladder is ruptured, trauma due to a penetrating body from without or to a bony fragment from within is responsible, and the rupture is usually extraperitoneal. Most instances of intraperitoneal rupture are due to the concussion of a blow at a time when the bladder is distended, and it is commonly the alcoholic who is the victim of this disaster. Spontaneous rupture occurs with relative rarity, and Stone (1931) could find only 40 cases in the literature. He found it to be due to disease of the bladder wall, inflammatory or malignant, or to obstruction at the bladder neck or urethra, leading to gross over-distension of the bladder. Paralytic lesions form a small percentage of this type. This spontaneous type of rupture may be extraperitoneal, though it is more often intraperitoneal, the former being associated with diseases of the bladder wall, whilst the latter most frequently occurs with an obstructive lesion.

In Stone's review the precipitating factor immediately responsible for the catastrophe was invariably a sudden increase in intra-abdominal pressure, due to such causes as attempted micturition, straining at stool, and sudden lifting of a heavy weight. On five occasions the distended bladder ruptured during labour and once during the manual expression of the placenta on the fifth day of the puerperium. Rupture has been reported by Sarkar (1944) and Mussnug (1947) in association with retention of urine due to an incarcerated gravid uterus. Claye (1941) describes a fatal case of rupture of the bladder wall on the tenth day of the puerperium due to a neglected distension with overflow, though in his case the tears were not complete and there was no extravasation of urine.

The mortality rate in this catastrophe has always been very high, and Stone's investigations showed that before 1900 the rates for intraperitoneal and extraperitoneal spontaneous rupture were 92.3% and 50% respectively. After 1900 the rates were 45.4% and *nil* respectively.

The clinical picture varies considerably, and the extra-peritoneal type, with its less acute symptoms, often does not reach the consultant until there are signs of extravasation of urine, with its concomitant infection. In the intraperitoneal type, however, after a period of retention of urine, a sudden increase in intra-abdominal pressure is followed by severe abdominal pain and collapse. The picture of "acute abdomen" with rigidity follows, though shifting dullness may also be elicited in many cases.

In most of the earlier cases treatment was unsatisfactory, as it was not realized that post-operative drainage of the bladder was essential. Since this has been carried out the results have steadily improved, and Stone maintains that repair of the rent in the bladder is unnecessary, so long as the peritoneal cavity is drained and a suprapubic cystostomy carried out.

Diagnosis of spontaneous rupture of the bladder is difficult unless there is a history of retention of urine, with an associated obstructive lesion. In the following case there was no suspicion of the true diagnosis until the peritoneal cavity was searched at laparotomy.

Case History

A married woman aged 36 had a normal delivery of her second child on September 25, 1950, after a labour lasting seven hours. Her doctor had to express the placenta, and she had a second-degree perineal tear, which was sutured. Her first child had been stillborn two and a half years before, following a severe pre-eclamptic toxæmia. During the puerperium the patient complained of abdominal discomfort, which was thought to be due to after-pains; but she was otherwise well, and her chart showed no rise of temperature. On the morning of the fifth day after delivery the midwife suggested that the time had come for her to spend a few hours a day lying on her abdomen lest her uterus should become retroverted. This injunction was obediently followed, and immediately the patient felt a severe pain in the upper abdomen and collapsed. She vomited once shortly afterwards.

I first saw her in consultation with the doctor a few hours later, when the above history was obtained. At this time her temperature was 97° F. (36.1° C.), the pulse rate was 106 and of fairly good volume, while she was sweating profusely. Her colour was good, however, and the diagnosis of pulmonary embolus, as suspected by the doctor, seemed improbable. The pain was localized to the upper abdomen, and on inspection the abdomen appeared generally distended, though this was most marked below the umbilicus. Palpation revealed a cystic swelling which filled the whole of the lower abdomen and rose to about two fingerbreadths above the level of the umbilicus. This was tender, but not as markedly so as the epigastrium and hypochondria. There was no rigidity, but the muscles had been stretched by the pregnancy and had not yet regained their tone, and the most outstanding feature was the pain felt when the pressure of the hand was released. The swelling in the abdomen was dull to percussion. A pelvic examination was not then carried out as it was obvious that the patient would have to be moved into hospital. A ruptured ovarian cyst was provisionally diagnosed, and arrangements were made for her transfer.

On arrival at hospital nearly two hours later, after an ambulance journey of 16 miles, she was immediately catheterized, 26 oz. (740 ml.) of rather dark-coloured urine being obtained. When she was seen immediately afterwards the clinical picture had changed. She looked more shocked than when first seen, with a cyanotic tinge about the lips, though the pulse rate was about the same, and the B.P. was 110/70. On examination, however, instead of the cystic lower abdominal swelling, there was now a

generalized distension of the abdomen, with dullness on percussion corresponding to that of an ascites instead of an ovarian cyst. In addition a fluid thrill could be elicited. Pain and tenderness were unabated, however, and laparotomy seemed imperative.

Half an hour later the patient was anaesthetized, though the induction was difficult owing to the vomiting of dark "coffee-ground" material. The stomach was emptied with a stomach-tube, after which there was less difficulty. A metal catheter was passed into the bladder and another 6 oz. (170 ml.) of clear urine obtained. On bimanual vaginal examination no pelvic abnormality could be found apart from the large involuting uterus. A fluid thrill was still clearly present, however.

The abdomen was opened by a midline subumbilical incision, and immediately a straw-coloured odourless fluid began to flow out. Six pints (3.4 litres) of this was removed with the sucker and the rest mopped out with gauze until a clear view of the abdominal contents could be obtained. The uterus was pale and normal in size for this stage of the puerperium, while the small bowel and omentum were also of normal appearance. Both ovaries and tubes were inspected and found to be normal. The upper abdomen was explored with a hand, and nothing abnormal was discovered. More by good fortune than design, the uterus was pushed backwards and the uterovesical pouch examined, and there in the fundus of the bladder was a hole big enough to admit one or two fingers; its edges were necrotic and gangrenous.

The necrotic edges were cut away and the hole was repaired by continuous sutures in two layers. An assistant introduced a Malecot self-retaining catheter into the bladder, via the urethra, before the abdomen was closed, to safeguard against further injury to the sutured fundus. A little remaining fluid was mopped out of the peritoneal cavity, and the abdomen closed without drainage. The patient's general condition was not very good, and an intravenous saline drip transfusion was started and a Ryle's tube left in the stomach. A pint (570 ml.) of blood was transfused a few hours later. The bladder was allowed to drain into a Winchester bottle. Penicillin, 200,000 units, was given three-hourly.

The post-operative course was impaired at first by a troublesome cough with a purulent sputum, though there was only slight pyrexia. Some intestinal distension developed, but spontaneous passage of flatus occurred on the third day, when the drip was discontinued. The Ryle's tube had been removed the day after operation. An enema was now given, and flatus resulted. By the fifth day the patient was eating a light diet, and sulphamezathine, 0.5 g. four-hourly, was started. The catheter was removed on the ninth day, and micturition was immediately possible, though in small quantities at a time. Estimation of residual urine in the morning and evening showed 25 oz. (710 ml.) in the bladder at first, though this gradually diminished to a few ounces by the fourteenth day, when further catheterization was considered unnecessary. Ambulation had been allowed as soon as the catheter was removed. Sulphamezathine was continued until the residual urine had disappeared. The patient was discharged from hospital on the eighteenth post-operative day.

Comment

Spontaneous intraperitoneal rupture of the bladder in the puerperium is an extremely rare event, and no record of any other case can be found, except one in which manual expression of the placenta was being attempted on the fifth day. There is therefore little to be learnt from such an occurrence, though it may be pertinent here to make the following remarks: (1) Palpation of the involuting uterus is still an important part of the after-care of the puerperal patient. Delay in the descent of the fundus is often an indication that the bladder is not

being emptied completely, and that residual urine is collecting. (2) Early ambulation is probably the best means of ensuring that a patient empties her bladder completely.

REFERENCES

- Claye, A. M. (1941). *J. Obstet. Gynaec. Brit. Emp.*, **48**, 235.
 Mussnug, G. (1947). *Geburts. u. Frauenheilk.*, **7**, 179.
 Sarkar, A. N. (1944). *J. Indian med. Ass.*, **13**, 113.
 Stone, E. (1931). *Arch. Surg., Chicago*, **23**, 129.

AUREOMYCIN AND CHLORAMPHENICOL IN CHANCROID

BY

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Reports concerning the action of "aureomycin" and chloramphenicol on chancroid have been somewhat conflicting. Thus, Wetherbee *et al.* (1949), who tested streptomycin, aureomycin, and a number of other antibiotics *in vitro* against *Haemophilus ducreyi*, thought that aureomycin had only a transitory effect, and that of the drugs tested only streptomycin was likely to be of value for soft sore. Nell and Hill, quoted by Robinson (1950), likewise found the inhibitory effects of aureomycin inferior to penicillin, but stated that chloramphenicol was effective. Working on the human infection, however, Zheutlin and Robinson (1950) reported three clinical cases successfully treated with 2 g. of aureomycin daily for 7–14 days, while Greenblatt *et al.* (1950) recorded yet another case. On the other hand, the latter workers believe that 5–10 g. of chloramphenicol is necessary.

The present work was undertaken in Africa during 1949, on both experimental and clinical infections.

Experimental Infection

An intradermal inoculation of 0.05–0.1 ml. of fluid aspirated from chancroidal buboes was given either to the patients themselves or to other volunteers. If both the donor and the recipient are untreated a local pustule develops within 48–96 hours and proceeds to chancroidal ulceration. If the recipient is treated with effective drugs at the time of inoculation, or the donor before inoculation, no such reaction occurs. Three series of experiments have been described (Willcox, 1950a, 1950b, 1950c), referring respectively to (1) the auto-inoculation of the donors; (2) the hetero-inoculation of volunteers treated with material from untreated donors; and (3) the hetero-inoculation of volunteers with material taken from treated donors 24 hours and later after the beginning of treatment.

Chloramphenicol.—Bubo fluid from Case 1 (see below), proved virulent by injection into an untreated volunteer, was injected into the patient himself, who then received 250 mg. of chloramphenicol orally three times daily for three days. A local bump was evident at 48 hours and persisted until 120 hours, when it was needled but found not to contain pus. Bubo fluid from this donor was also inoculated into five other men 24–48 hours after the treatment had begun, and all failed to show a take, thus indicating that the fluid had lost its virulence. Four others, who received inoculations with